III.—THE VAGUS NERVE.

I.—Die trophischen Beziehungen der Nervi Vagi zum Herzmuskel. Von Dr. Hermann Eichhorst. Berlin: 1879. A. Hirschwald. Pp. 38. (*The trophic relations of the vagi nerves to the heart muscle.*)

II.—Folgen der vagus-durchschneidung bei Vögeln. (*Consequences of Section of the vagus in birds.*) Richard Zander. *Pflüger's Archiv für Physiologie*, Bd. 19, p. 263 (1879).

I. Up to the present day it is still an open question, in what manner death, the inevitable consequence, is caused by section of the pneumogastric nerves. The autopsy of the animals reveals almost always an inflammation of the lungs. But since we know, from the researches of Traube and others, that this condition is caused by the entrance of saliva, food, etc., into the insensible bronchial tubes, we can prevent it altogether by proper management. Nevertheless, the animals operated upon cannot escape death. As the true cause of death, Eichhorst claims an impaired nutrition of the heart, and bases this view upon the following observation, commencing with birds, but studying also the phenomena in mammals.

It has been claimed by Cl. Bernard that irritation of the vagus *in birds* does *not* arrest the heart. While some other observers had previously arrived at other results, the matter was particularly investigated by Einbrodt (*Müller's Archiv*, 1859, p. 439), who *did* prove an inhibitory influence of the vagus upon the heart of the bird, but found the nerve easily exhaustible. This result Eichhorst confirms fully, but he disagrees with the former author as to the cause of death. Einbrodt stated that his animals died from impaired digestion and nutrition, while Eichhorst claims that these conditions can be entirely prevented by proper care. But nowhere in the brochure does he tell us in what the care consists.

The consequences of division of the two vagi in pigeons, ravens and jackdaws are the following: The pulse is at once accelerated, and respiration becomes dyspnœic. Inspiration is deep and slow, while the expiratory effort is rather sudden, and accompanied by a squeaking sound. The respiratory movements become reduced to 20, 10, or even 5 per minute. The pulse becomes too rapid to be counted, amounting to more than 250 per minute, and the cardiac sounds lose their clear timbre and become muffled. Various other symptoms occur, but they are neither constant nor important. Attempts at swallowing and oozing of a viscid fluid from the mouth are often observed for a

short time after the operation. *Contrary to all experience upon mammals, Eichhorst claims to have found no impairment of digestion in birds.* He claims to have seen them eat, and found their stomach and intestines filled—food undergoing digestion.

The disturbances of circulation and respiration diminish gradually during the course of the second day after the operation. Still, the animals are evidently sick. Their eyes appear dull, their plumage is disordered, the birds are crouched in a corner of the cage in an apathetic manner. But in a few days they regain their former liveliness, and present no appearance of suffering. Nevertheless, they die inevitably, usually about the end of the first week. The death occurs sometimes quite suddenly; in other instances it is preceded by dyspnœa for some hours.

After death no change can often be detected with the unaided eye. The author denies directly that the birds presented any appearances of starvation. *In birds pneumonia does not occur as the consequence of section of the vagi.* Occasionally a slight quantity of a clear, frothy fluid can be pressed out of the pulmonary tissue, but there is never any evidence of inflammation. This is in accordance with Traube's theory of the origin of the pneumonia from swallowing the buccal fluid and food, since this cannot occur in birds.

The heart is always found diseased after death. Sometimes no morbid change can be seen with the unaided eye; in other cases the organ is pale, relaxed and friable; most frequently it is studded with yellowish streaks and spots of varying extent—mostly near the inner surface. The left ventricle is always more implicated than the rest of the heart. The extent of the change does not depend alone upon the time after the operation, but varies in different animals. The microscope reveals always that a large number of the muscular fibres of the heart are filled with fat granules and drops, and that the striation has disappeared in the entire fibre, or at least a portion of it. The muscle nuclei can no longer be recognized.

This fatty degeneration might be referred to three conditions: the dyspnœa, the pulse acceleration, or the division of any trophic nerve fibres. The first ground, suggested by the recent researches of Fränkel on fatty degeneration as the consequence of reduced supply of oxygen, is really untenable. Previous observers have found that (at least, in rabbits), the absolute quantities of O and CO_2 remain the same as before section of the pneumogastric nerves, the disturbance of respiration referring only to the rhythm. For want of apparatus, the author could not verify this in the case of birds; but he refers at least to the perfect return of normal respiratory rhythm in the course of the second day.

The degeneration can also not be attributed to the increased work of the heart. In order to exclude this as the cause Eichhorst supplied birds during a period of even ten days with suffi-

cient atropin to paralyze the inhibitory fibres and raise the pulse to 300 to 400 per minute. Nevertheless no degeneration of the heart muscle was produced. Hence the author suggests the existence of trophic fibres in the trunk of the vagus as the real cause.

In rabbits the cardiac changes are not seen ordinarily on account of the early death of the animals. They are usually carried off within the first twelve hours by a pneumonia caused by the entrance of food and fluid into the lungs. But the pulmonary process can be prevented by tying a canula into the opened trachea, and in this case the rabbits can survive two or four days. Taking these precautions Eichhorst could always find cardiac degeneration. The heart was soft, pale and friable, and as in the case of the birds, marked with irregular fine, yellow streaks. Many or most fibres had lost their striation, and presented the appearance of *waxy* degeneration. In birds, on the other hand, only *fatty* degeneration could be seen, there was no proliferation of nuclei, or any other evidence of an inflammatory process.

In these animals Eichhorst could also satisfy himself, that the lesion was not due to overwork, since a pulse-acceleration produced by the administration of atropin for a fortnight had no such result. The inhibitory fibres are all derived from the spinal accessory nerve. Destruction of this nerve accelerates hence the pulse as much as section of the vagi, but does not cause any cardiac lesion.

The same results were obtained in dogs. The cardiac degeneration in these animals is rather of the fatty type, but remarkably slight in extent, even if the death is postponed by the avoidance of pulmonary troubles. The difference in the intensity of the degenerative process in different animals, Eichhorst attributes to the different structure of the cardiac muscle. In dogs the fibres of the heart muscle are tough and relatively thick, while in birds they represent much more delicate structures.

The main conclusions which Eichhorst bases on his experiments are: *That the vagus contains fibres, the division of which leads to fatty degeneration of the heart-muscle, and that this condition is the cause of death.*

There is, however, nothing convincing in the author's reasoning. There is no proof whatever, except by a questionable exclusion, that death is really due to this state of the heart. No exact account of the mode of death, no graphic curves of the failing circulation can be found in the book. The author denies that the animals die of starvation, and this indeed seems improbable on account of the very short time between the operation and the fatal issue. But may not the failure of general nutrition, which all other observers *have* found, cause the cardiac degeneration, especially if we take into account the overwork of the heart? At any rate both Manassein and Falk have found the cardiac degeneration in starved animals. The existence of true trophic fibres in the vagus is only a *possible* or even *plausible* explana-

tion of the author's observations, but that it is the *true* explanation he fails to prove. His entire work, valuable as it is, suggests rather new problems, than positive answers to previous ones. Eichhorst's results have induced Wassilijew to take up the same subject in Botkin's laboratory in St. Petersburg. (*St. Petersburger med. Wochenschrift*, Nos. 7 and 17, 1879.)

He found likewise a cardiac degeneration after section of the vagi in the rabbit, but attributes it to the starving condition. He obtained also the same lesions by inducing an irritative process in the nerve by pricking it. But in reality he induced by this supposed "irritation" an atrophy of many fibres of the vagus, as is evident from his microscopic examination. Hence, his conclusion as to the causation of the cardiac lesion by a continued state of irritation of the vagus, is not tenable.

II. The article of Zander impresses the reader at once as the thorough work of a good observer. Contrary to the results of previous observers, including Eichhorst, he found that section of the two vagus nerves in birds does not leave the lungs intact. In every instance the operation was followed by disseminated spots of hyperæmia and œdema of the lungs. In mammals on the other hand the nerve section causes a true pneumonia, which is mainly or wholly due to the entrance of fluid and food into the trachea. In birds this does not occur, since their upper larynx is supplied with sensibility and motility by the *superior* laryngeal nerve, which is not affected by the division of the vagus in the neck. But on dividing these nerves also, the contents of the gizzard, putrid on account of the paralysis of that organ, enter the larynx, and the result is a septic pneumonia. The same can be produced in birds in which the vagi alone have been divided, on injecting artificially such putrid fluid into the lungs.

The pulmonary hyperæmia following nerve section is more considerable immediately after the operation than a few days later. Hence, Zander considered it likely that this was due to an irritation of vaso-dilator nerves contained in the vagus, by the act of division. On laying bare the lung he did actually find hyperæmia of the lung on dividing the vagus, which gradually diminished, but could be reproduced by irritating the distal nerve end. Corresponding observations were made on the temperature of the lungs, as measured by means of thermo-electric needles. Division or irritation of the vagus increases the temperature of the corresponding lung for some hours. If both nerves are divided, the temperature in the rectum sinks a few degrees—no doubt on account of the greater loss of heat from pulmonary hyperæmia—but within some hours it rises again to nearly the normal height, and remains constant until death. If the contents of the gizzard are prevented from entering the lungs, the pulmonary changes are too slight to cause death. What is, hence, the real cause of the death?

Young birds die mostly from suffocation. Their larynx is not

yet ossified, and becomes compressed, like a valve during inspiration, on account of the paralysis of the dilating muscles.

In other cases death occurred quite rapidly, without any preceding symptoms, and Zander attributes it, hence, to sudden paralysis of the heart; but this explanation is not satisfactory.

In adult animals it is even more difficult to find the real cause of death. In some instances Zander found a fatty degeneration of the heart. In other cases the post-mortem appearances are detailed, without any mention whatever of this condition. It seems that Zander did not use the microscope to any great extent. He admits, however, that this degeneration may cause death by cardiac paralysis. Nevertheless, the heart thus degenerated was found beating in some birds after failure of the respiration! The existence of trophic nerves, however, the author denies altogether, attributing this fatty degeneration to the state of inanition caused by vagus-section. At any rate, he claims to have found the same morbid process in the hearts of starving pigeons. The most important cause of death Zander seeks in the starvation due to paralysis of the gizzard and œsophagus. Although the animals can eat, food is of no use to them, since it is all retained in the gizzard, and putrifies in its cavity. It cannot even be pushed by force into the muscular stomach. The animals dying about the end of the first week presented the usual post-mortem appearances of death from starvation. Some attempts made by the author to maintain life by artificial feeding did not succeed.

Notwithstanding the care betrayed in the paper, as well as the many new facts found in it, the cause of death after vagus-section is not quite clear. As it appears from the accounts of his experiments, the animals died often before they had lost forty per cent. of their weight, a loss found regularly by Chossat. They died, besides, within a period of time rather too short to permit death by starvation. H. G.

IV.—RICHET: CEREBRAL CONVOLUTIONS.

STRUCTURE DES CIRCONVOLUTIONS CEREBRALES (ANATOMIE ET PHYSIOLOGIE). THESE PRESENTEE ET SOUTENUE A LA FACULTE DE MEDICINE DE PARIS POUR LE CONCOURS DE L'AGREGATION (SECTION D' ANATOMIE ET DE PHYSIOLOGIE). Par le Dr. Charles Richet. (*Structure of the Cerebral Convolutions.*) Paris, 1878. 172 pages. 2 litho. plates.

The first part of this memoir is devoted to a description, very fairly minute and accurate, of the general structural anatomy of the cerebral cortex, the descriptive anatomy of the convolutions being left to the later portion of the work. While it contains nothing particularly original, it is yet a very good *resumé*